finer distinctions to be made in the pitch and quality of the percussion notes, so that greater accuracy is secured in the percussion of the chest and its contents. He states that very light percussion strokes must be used—preferably the so-called "threshold percussion"—and the patient examined in a very quiet room. The results obtained by this method of percussion in his hands are exceedingly accurate. The instrument is small and can be folded to carry in the pocket, and is so simple in design that any clever mechanic can construct one in a very short time.

Diminished Blood Platelets and Marrow Insufficiency.—Minot (*Arch. Int. Med.*, 1917, xix, No. 6, p. 1062) discusses at length the findings in a series of cases of purpura hemorrhagica and aplastic anemia. The two conditions are often confused, and in most text-books no differential diagnosis between the two conditions is mentioned. There is also often much confusion between the chronic types of purpura hemorrhagica and hemophilia, this confusion usually arising from incomplete studies of the blood in these cases. Minot's conclusions were derived from the literature (of which he presents an extensive bibliography) and from the study of some 25 cases in the purpura hemorrhagica and aplastic anemia groups. Purpura hemorrhagica is a condition in which there is bleeding from one or more mucous membranes, often purpuric skin lesions, associated with diminished blood platelets, a delayed bleeding time, and a soft non-retractile blood clot. The coagulation time is normal or slightly delayed, rarely much delayed. The condition is more usually secondary to some recognized disease, as diphtheria or tuberculosis, and is also to be considered secondary when it appears as a symptom accompanying aplastic anemia, leukemia, bone-marrow tumors, pernicious or splenic anemia, etc. When no recognized cause can be found for its presence it is then spoken of as a disease entity—purpura hemorrhagica or Werlhof's disease. Idiopathic purpura hemorrhagica may be a disease that is acute, subacute, chronic or of an intermittent nature. A congenital and hereditary type exists. Acute aplastic anemia is a disease of unknown etiology which runs a progressively downward, fatal course of three to six weeks' duration. It occurs usually in patients between fifteen and thirty years of age. Fever frequently occurs, and there is no evidence of increased blood destruction as in pernicious anemia. It seems that whatever the cause of the disease may be its action is to inhibit the activity of the blood forming elements in the bone marrow, for at autopsy the marrow is found to be completely fatty. Near the termination of pernicious anemia, and in the course of certain infections, as sepsis, malignant endocarditis, typhoid fever, diphtheria, miliary tuberculosis, etc., aplasia or exhaustion of marrow may occur. In such instances we speak of a secondary aplastic anemia, the only difference between this form and the idiopathic aplastic anemia being that in the first type we recognize a source for the toxin formation and in the second we do not. In aplastic anemia the blood picture shows no evidence, or very little, of regeneration of the red cells, platelets or polynuclear leukocytes. The red cells may be very low in number (often 500,000 before death), and yet the color index averages 0.8 or slightly higher, and there is little or no variation in shape and very little variation in size. Polychromatophilia, stippling, blasts,

reticulated red cells and Howell-Jolly bodies are absent or rare. If the color index is high, and there is an occasional and unduly large and especially an oval shaped erythrocyte found, with all the other characteristics of an aplastic anemia, the suspicion that the case was one in which an aplasia of the bone marrow had developed in the course of pernicious anemia is warranted. The platelets in aplastic anemia are markedly diminished in number, often nearly absent, and they vary in size in different cases. The white cells are diminished in number, more so as the disease progresses. The leukopenia consists in an absolute diminution in the number of polynuclear cells and usually disappearance of the eosinophiles, with thus a relative but not an absolute increase in the lymphocytes, the percentage of lymphocytes averaging 73 per cent. These cells are of the normal type. There is usually a "shift to the left" (Arneth) in the polynuclear types contrasted with the frequent "right handed shift" pointed out in pernicious anemia by Briggs. The only distinctive feature of the blood picture in purpura hemorrhagica is the marked decrease in the number of blood platelets. The blood picture in this disease is therefore one consistent with a bone marrow which is incapable of forming platelets, but competent to a greater or less degree to produce polynuclears and red cells. The anemia in these cases is due to the hemorrhages and not apparently to any lack of formation or increased destruction of red cells. The red cells then are of the type usually found in an acute or chronic posthemorrhagic anemia—variation in size evident in shape, slight, some achromia occurs, and the color index is usually lowered. Polychromatophilia and stippling occur, occasionally a few normoblasts, and rarely a Howel-Jolly body. Reticulated red cells are increased when definite anemia is present. The white cells are usually increased, usually about 12,000 and sometimes higher, with an increased percentage of polynuclear elements in the higher, and often in the lower counts. There seems to be a slight "left-handed shift" (Arneth) in the types of polynuclears. The platelets are diminished often as low as 1000. The normal count is 225,000 to 325,000. When the platelets are decreased below 60,000 hemorrhages tend to occur. This decrease in platelets is in marked contrast to the increase, frequently to 1,000,000, seen in ordinary posthemorrhagic anemias in which the picture is one of active bone marrow regeneration of all three of the formed elements. The platelets usually vary greatly in size being both abnormally small, and abnormally large. There are also cases which present blood pictures intermediate between these two groups, and which suggest that in them some agent is at work interfering unequally with the production of the formed elements. Such cases are those of purpura hemorrhagica which show no increased white count or a leukopenia, or a normal or slightly increased white count with a moderate increase in lymphocytes (50 per cent.). Other cases show chronic anemia without evidence of blood destruction and little hemorrhage, red cells suggestive of poor erythrocytic activity of the marrow, leukopenia with slight relative and absolute decrease of the polynuclears, and somewhat diminished platelet counts. These cases suggest a stage midway between purpura hemorrhagica and aplastic anemia. There are also cases in which the picture is that of an aplastic anemia, and yet in which the red cells show evidence of regeneration and the leukopenia and decrease in platelet count are not marked. These intermediate

cases seem related to the more clearly cut disease known as idiopathic purpura hemorrhagica because in common with it the nature of the disease process is unknown, the most marked symptom is purpura, and the most striking blood finding is the diminution in blood platelets. The data suggest that all these conditions with diminished platelets and purpura are similar, whether the marrow only is involved or whether there is increased platelet destruction in the plasma. The stimulation of the marrow in these cases offers the greatest hope in their treatment, and this is best done by transfusion. The study of the absolute and relative numbers of the polynuclears, the reticulated red cells and the platelets is the most satisfactory way of determining bone marrow activity. Needless to say, in aplastic anemia there is no evidence of bone marrow activity following transfusion, but in some cases of purpura hemorrhagica all of the formed elements may be favorably affected after transfusion, and in other cases only the red cells and polynuclears. In the intermediate cases the beneficial effects of transfusion upon the red and white cell production may be very slight, suggesting a partial aplasia of these marrow elements. The studies of the bone marrow in idiopathic purpura hemorrhagica have been rather few, and Minot reports an increase of the number of megakaryocytes in the marrow of a boy, aged fourteen years, who died from idiopathic purpura. The platelets may be reduced in the peripheral blood by destruction of the megakaryocytes of the bone marrow, by some toxin which inhibits the formation of platelets by the megakaryocytes or by destruction of the platelets in the peripheral circulation. It may possibly be that rapid destruction of platelets in the peripheral circulation causes in some cases, a depression of the whole marrow. In the aplastic anemias the reduction of platelets is due to diminution of the megakaryocytes just as the decreased red count is due to diminution in the red and white cell elements of the marrow. In certain of these cases presenting the blood picture of aplastic anemia the marrow may show areas of aplasia and areas of hyperplasia. These cases are really ones of incomplete aplasia and many cases terminating as complete aplasias show early in their courses a few regenerative red cells, produced by foci of still active marrow attempting to repair the destructive process. In the symptomatic cases of purpura hemorrhagica appearing in leukemia or tumor infiltrations of the marrow the megakaryocytes have been displaced by the new cells, so that they are decreased in number. In aplastic anemia it is reasonable to assume that some factor attacks the whole marrow, the red cell elements being involved first and the platelets later as evidenced by the hemorrhage appearing after the anemia, while in purpura hemorrhagica the platelets are attacked first so that hemorrhages appear before the anemia which is largely referable to the loss of blood. In the cases intermediate between purpura hemorrhagica and aplastic anemia the "toxin" is probably such that, besides injuring the activity of the megakaryocytes, it impairs the function of either or both of the red and white cell elements of the marrow. In chronic diseases where depression of the marrow exists, there seems to be no "toxin" at work, but possibly a congenital peculiarity of the blood forming elements of the marrow, or a supernormal sensitivity to some toxin that ordinarily produces no such effect. It is also conceivable that disease of some other organ such as

the spleen may cause depression or inactivity of the bone marrow, just as disease of one of the glands of internal secretion may depress one of the others. The author then discusses the differential diagnosis of purpura hemorrhagica and aplastic anemia, pointing out that drug purpuras Henoch's purpura and Schoenlein's disease are not true purpura hemorrhagica. Purpura hemorrhagica and aplastic anemia must not be confused with hemophilia, the non-leukemic phase of leukemia, bone marrow tumors, pernicious anemia and splenic anemia. The distinguishing characteristics of hemophilia are the normal platelet count, blood clot and bleeding time, with the greatly delayed coagulation time; of the non-leukemic phase of leukemia, the picture of active regeneration of the red cells and polynuclears, the presence of abnormal forms of lymphocytes, and swelling of the lymph nodes; of bone marrow tumors, the history, physical examination, roentgen-ray findings (Bruce Jones protein in the urine in myeloma) and presence of abnormal cells, plasma cells or tumor cells should they occur in the peripheral blood; of pernicious anemia after aplasia of the marrow has occurred, the history of remissions, sore tongue and spinal involvement, color of the skin, and a high color index of the red cells with the presence of occasional large and abnormally shaped red cells; of splenic anemia, rather marked enlargement of spleen and later of the liver, though this disease is often difficult to distinguish from purpura hemorrhagica. The author gives a short classification of the types of purpura hemorrhagica and aplastic anemia and suggests that, instead of calling a group of disease conditions "purpura hemorrhagica," we use more specific terms and referred to cases of "insufficiency of the marrow with especial involvement of the platelets or other formed elements in varying degrees."

SURGERY

UNDER THE CHARGE OF

T. TURNER THOMAS, M.D.,

ASSOCIATE PROFESSOR OF APPLIED ANATOMY AND ASSOCIATE IN SURGERY IN THE UNIVERSITY OF PENNSYLVANIA; SURGEON TO THE PHILADELPHIA GENERAL HOSPITAL AND ASSISTANT SURGEON TO THE UNIVERSITY HOSPITAL.

Treatment of Gunshot Wounds of the Abdomen, with a Summary of 500 Cases seen in an Advanced Casualty Clearing Station.—Lockwood, Kennedy and MacFie (*British Med. Jour.*, March 10, 1917, p. 317) say that the earlier the patient could be operated on the better were the results. As late as twenty hours after being wounded, operation was considered the best course, and their results justified them. Judgment in cases seen after twenty to thirty hours presented much greater difficulties; there was always the danger of increasing the damage by manipulation during the operation. If general plastic peritonitis had developed, interference was not only valueless but dangerous; they were content to insert a pelvic drain, or, if a fecal fistula was found, to mop it out carefully and drain.